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ORIGINAL ARTICLE

Prevalence of comorbidities in obese New Zealand children and adolescents at enrolment in a community-based obesity programme

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Aim: The aim of this study was to describe the characteristics at enrolment of children and adolescents referred to an obesity programme and to determine how the prevalence of comorbidities differed in Indigenous versus non-Indigenous children.

Methods: Participants were residents of a semi-rural region of New Zealand (NZ). Eligibility was defined by a body mass index (BMI) of ≥98th percentile or >91st centile with weight-related comorbidities. Fasting blood, medical and physical assessments were obtained.

Results: During the recruitment period from January 2012 to August 2014, 239 participants, aged 4.8–16.8 years, undertook assessment. Average BMI standard deviation score was 3.09 (standard deviation (SD) = 0.60, range 1.52–5.34 SD). The majority of participants were of either Maori (NZ's indigenous people (45%)) or NZ European (45%) ethnicity; 29% of participants were from the most deprived quintile of household deprivation.

Maori participants were more likely than NZ Europeans to have a mother who smoked during pregnancy (52% vs. 28%, $P = 0.001$), a family history of type 2 diabetes (66% vs. 53%, $P = 0.04$), acanthosis nigricans on examination (58% vs. 20%, $P < 0.0001$), a low serum high-density lipoprotein cholesterol (27% vs. 14%, $P = 0.03$) or high serum triglyceride (38% vs. 24%, $P = 0.03$) concentration.

Conclusion: The unique aspect of this study was the ability to recruit high levels of Maori participants and those from most deprived areas, indicating a high level of acceptability for these target groups. Comorbidities were prevalent in this cohort of overweight/obese school-aged children. While there were some differences in comorbidity prevalence between Maori and NZ Europeans, the overall clinical picture in our cohort, irrespective of ethnicity, was of concern.

Key words: adolescent; comorbidity; Indigenous population; New Zealand; pediatric obesity.

What is already known on this topic

1 Child and adolescent obesity rates in New Zealand are high compared with other countries internationally.
2 Rates of child and adolescent obesity are worse in Maori (New Zealand's Indigenous population) and those from most deprived backgrounds.
3 Comorbidities in obese children and adolescents exist and emphasise the need to reduce the future burden of this non-communicable disease.

What this paper adds

1 Obesity programmes that address accessibility and acceptability for Indigenous people and those from most deprived backgrounds can lead to improved participation rates with these groups.
2 Irrespective of ethnicity, concerning levels of comorbidities are prevalent in obese New Zealand children and adolescents.
3 Maternal smoking rates in pregnancy, especially in Maori, are particularly noteworthy.

Internationally, the combined prevalence of overweight and obesity in children has increased by 47% from 1980 to 2013.[1] The implications of the burden of non-communicable diseases associated with obesity and the impact on health systems are of global concern.[2] As a result, the World Health Organization (WHO) has now released recommendations to end childhood obesity.[3]

The prevalence of overweight and obesity in New Zealand (NZ) adults is the third highest in Organisation for Economic Co-operation and Development (OECD) countries.[4] Among 2- to 14-year-old children, the prevalence of obesity (as defined by International Obesity Task Force BMI reference values) has

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increased from 8.4% in 2006/2007 to 10.8% in 2014/2015.[5] Maori (NZ's Indigenous people) children are 1.6 times more likely than non-Maori children to be obese.[5] As childhood obesity often leads to adult obesity,[6] these recent trends raise additional concerns about the future burden of obesity-related disease in our adult population. Weight-related comorbidities track from childhood into adulthood and have been well documented.[6]

Marked health disparities exist between Indigenous and non-Indigenous groups globally, and where information has been collected, these disparities are also evident in relation to childhood obesity.[7] The need to address the worsening obesity epidemic and to eliminate the disparities in obesity-related health outcomes between Indigenous and non-Indigenous groups is one of NZ's highest priority public health issues.[8] There are no data that we are aware of examining the differential effect of obesity on comorbidity in Maori and NZ European (NZE) children. Our primary objective, in the reporting of data contained in this manuscript, was to describe the baseline medical characteristics (prior to randomisation) of children and adolescents referred to the 'Whanau Pakari' multidisciplinary intervention trial (targeting Maori, in particular) and to describe the obesity-related comorbidities present at enrolment. Our secondary objective was to determine whether the prevalence of obesity-related morbidity differed between Indigenous and non-Indigenous children and adolescents.

Methods

Whanau Pakari is a community-based obesity programme currently being conducted in Taranaki, a semi-rural region of NZ with a population of 23 139 children aged 0–15 years, of which 81% identify as NZE, 28% as Maori and 1% as other ethnicity.[9] The methodology of Whanau Pakari has been described elsewhere.[10] Essentially, Taranaki children and adolescents were eligible if aged from 5 to 16 years and were either obese or overweight with significant weight-related comorbidities. Accessibility and appropriateness of the service were key to our approach in targeting at-risk groups. For example, we adopted strategies to remove barriers to access, such as providing a home-visit model and 'de-medicalising' the assessment. In addition, barriers and facilitators to engagement were considered in service and trial design.[10,11] We defined obesity and overweight using a modification of the United Kingdom Cole definitions of body mass index (BMI) ≥98th centile and >91st centile, respectively.[12]

We obtained ethical approval from the NZ Health and Disability Ethics Committee (CEN/11/09/054) and written and verbal informed consent from participants or their guardians. The trial was registered with the Australian NZ Clinical Trials Registry (ANZCTR: 12611000862943).

Data collection

Two hundred and thirty-nine baseline assessments were undertaken in the participants' homes. These assessments included a focussed medical and physical review, with requests for fasting blood samples from each participant (clear instructions were provided regarding fasting for at least 8 h prior to the blood test). A proxy report of medical history was obtained from an adult family member.

Measures

Height was measured to 0.1 cm using the average of three readings on a Seca 213 portable stadiometer (Seca, Hamburg, Germany). Weight was measured to 0.1 kg using Seca 813 digital scales. Waist circumference (WC) was measured using a Seca 201 standard measuring tape (at mid-point between the lower margins of the rib and the top of the iliac crest to 0.1 cm at the end of normal expiration).[13] Coefficient of variance analysis of WC was assessed, with a confirmed intra-user variability of 0.04%. Waist–height ratio (WHtR) was calculated from WC and height. Hip circumference was measured as the widest girth. We calculated BMI and BMI standard deviation score (SDS) using United Kingdom Cole normative data[14] on the uploadable KIGS auxology software (Pfizer Endocrine Care TM, New York, NY, USA). Height percentiles were calculated using gender-specific growth charts for 2- to 18-year-olds recommended by the Australasian Paediatric Endocrine Group,[15] based on Centers for Disease Control data.[16] Pubertal status by self-report was attained using Tanner staging pictures on the gender-specific growth charts. Accompanying adult BMI was measured where consented to, using the height and weight methods described above.

Casual blood pressure (BP) was measured in the home with aneroid sphygmomanometry, using a Welch Allyn sphygmomanometer and size-appropriate cuffs. If abnormal, the BP was repeated two further times during the 1–1.5 h assessment to reduce the possibility of white-coat hypertension. If initially elevated with reduction at subsequent measures, the lowest BP measurement was recorded. To improve accuracy, we calculated BP SDS using an age-based paediatric BP reference chart calculator.[17] SDS scores were then converted to percentiles. Prehypertension was defined as systolic and/or diastolic BPs between the 90th and 95th percentile for gender, height and age.[18] Hypertension was defined as systolic and/or diastolic BP >95th percentile.

Investigations

Low-sensitivity C-reactive protein (CRP), liver function tests, glycated haemoglobin (HbA1c) and fasting glucose, insulin and lipids were performed on an Abbott Architect c8000 chemical analyser (Abbott Laboratories, IL, USA) with coefficient of variances (CVs) between 1.6 and 11.2%. Insulin levels were measured by immunoassay on a Roche Cobas e411 analyser (Roche, Basel, Switzerland) with a CV of 4.1%. We determined the presence of raised CRP, abnormal fasting glucose, raised glycated haemoglobin, elevated fasting insulin level (>80 pmol/L based on adult upper limit of normal), abnormal fasting lipids and abnormal liver function tests.

Data analyses

Categorical variables were compared using χ^2 and Fisher's exact tests. Associations with outcomes of interest were examined using binary logistic regression models, accounting for important confounding factors: age, gender, ethnicity and pubertal status (as well as WHtR where it was necessary to adjust for adiposity).

For continuous outcomes, multivariable linear regression models were used, accounting for the above-described confounders. Secondary analyses were carried out comparing data between Maori and NZE. Parameters associated with glycaemic control and liver functions, as well as CRP values, were log-transformed to approximate normality. Data were analysed using Minitab (v.16, Pennsylvania State University, State College, PA, USA) and SAS v.9.3 (SAS Institute, Cary, NC, USA). All statistical tests were two-tailed, with significance maintained at $P < 0.05$. Where appropriate, data provided in the text are means and standard deviations (SD) or odds ratios (OR), with associated 95% confidence intervals in brackets.

Results

Demographics

Table 1 summarises the baseline demographic characteristics of the 239 participants assessed within the service from January 2012 to August 2014 (age range 4.8–16.8 years). Participants were predominantly of Maori (45%) or NZE (45%) ethnicity, with the remainder being of Pacific (3%), Asian (3%) or other ethnicities (4%). Twenty-nine percent resided in the most deprived quintile of NZ households (compared with 15% of the population of Taranaki).[20] Of the 239 participants, nine (4%) had a BMI at the 98th percentile, and 224 (94%) had a BMI >98th percentile on entry. Six (2%) participants entered with a BMI between the 91st and 98th percentile with weight-related comorbidities; all had a family history of weight problems and/or type 2 diabetes: one patient had type 1 diabetes, and another patient had pre-diagnosed type 2 diabetes. This group ($n = 6$) were excluded from all analyses relating to family history and glycaemic control, liver function, lipids and CRP.

Table 1 Characteristics of the 239 study participants at recruitment

Female gender (%)	125 (52)
Age in years, mean (SD)	10.7 (3.2)
Ethnicity†	
Maori (%)	109 (45)
New Zealand European (%)	108 (45)
Pacific (%)	6 (3)
Asian (%)	6 (3)
Other (%)	10 (4)
Deprivation index (quintiles)‡	
1 – least deprived (%)	23 (10)
2 (%)	38 (16)
3 (%)	48 (20)
4 (%)	60 (25)
5 – most deprived (%)	70 (29)
Accompanying adult	
Mother (%)	194 (81)
Father (%)	29 (12)
Other (%)	16 (7)
BMI (kg/m^2), mean (SD)§	33.6 (7.9)
BMI ≥30 kg/m^2 – obese (%)§	155 (68)
Living arrangements¶	
Two-parent household (%)	121 (51)
One-parent household – mother (%)	91 (38)
One-parent household – father (%)	10 (4)
Extended family (%)	11 (5)
Non-family caregiver (%)	3 (1)
Unknown (%)	3 (1)

†Prioritised ethnic group. ‡Quintiles of level of household deprivation based on the NZ Deprivation Index 2006.[19] §Parameter was measured where consented to ($n = 228$), otherwise not included. ¶Not stated for three. BMI, body mass index.

Medical history

Table 2 summarises the medical history of participants. Based on proxy reports, almost one-third had difficulty getting to sleep ($n = 76$, 32%). Snoring more than half the time (i.e. four or more nights per week) was found in half of participants ($n = 119$, 50%), with witnessed breathing pauses in one-fifth of the cohort ($n = 48$, 20%). A television, computer or device was present in the bedroom of almost half ($n = 111$, 46%) of the children. After adjustment for adiposity, a larger proportion of children who had

Table 2 Medical history of study participants at recruitment

	Subjects (*n*)	Value
Weight concern		
Age at first concern (years), mean (SD)	202	6.6 (3.1)
Duration (years), mean (SD)	203	4.1 (2.7)
Headaches (%)	239	77 (32)
Visual disturbance (%)	239	55 (23)
Diagnosed conditions		
Type 1 diabetes (%)	239	1 (0.4)
Type 2 diabetes (%)	239	1 (0.4)
Asthma (%)	239	59 (25)
Sleep		
Difficulty getting to sleep (%)	239	76 (32)
Television/computer in bedroom (%)	239	111 (46)
Hours per night, mean (SD)	239	10.2 (1.0)
Insufficient sleep (%)†	239	8 (3)
Snoring more than half the time (%)	239	119 (50)
Witnessed breathing pauses (%)	239	48 (20)
Diagnosed obstructive sleep apnoea (%)	239	9 (4)
Birth and development		
Smoking during pregnancy (%)	223	88 (40)
Gestational age (weeks), mean (SD)	232	39.4 (2.3)
Birthweight (kg), mean (SD)	230	3.46 (0.72)
Birthweight SDS, mean (SD)‡	227	0.20 (1.36)
Developmental concerns (%)	239	21 (9)
Family history§		
Type 1 diabetes (%)	233	31 (13)
Type 2 diabetes (%)	233	143 (61)
Weight problems (%)	233	189 (81)
Early obesity (%)	233	25 (11)

†Lower limits as recommended by the National Sleep Foundation: <10 h (aged 3–5 years), <9 h (6–13 years) and <8 h (14–17 years).[21] ‡Based on Niklasson Swedish reference data.[22] §Excluding six participants with BMI in the 91st to 98th percentiles who had weight-related comorbidities. SDS, standard deviation score.

Table 3 Examination findings of participants at recruitment

Examination finding	Total, *n* = 239
BMI SDS, mean (SD)	3.09 (0.60)
Waist circumference (cm), mean (SD)†	86.4 (14.1)
Waist height ratio, mean (SD)	0.59 (0.06)
Waist height ratio > 0.5 (%)	223 (94)
Acanthosis nigricans (%)	98 (41)
Prepubertal (%)‡	114 (48)
Pre-hypertension (%)§	17 (7)
Hypertension (%)¶	9 (4)

†At mid-point between the lower margins of the rib and the top of the iliac crest to 0.1 cm at end of normal expiration.[13] ‡Based on self-report. §Systolic and/or diastolic blood pressure between 90th and 95th percentile. ¶Systolic and/or diastolic blood pressure >95th percentile, based on fourth report percentiles,[18] calculated using blood pressure SDS.[17] BMI, body mass index; SDS, standard deviation score.

Table 4 Laboratory markers of disease in study participants at recruitment

	Samples, *n*	Abnormal, *n* (%)
Inflammatory marker†		
ls-CRP 1.0–3.0 mg/L	183	92 (50)
ls-CRP >3.0 mg/L <15.0	183	45 (25)
ls-CRP >15 mg/L	183	6 (3)
Glycaemic control‡		
Fasting glucose >7 mmol/L	179	1 (0.6)
HbA1c >42 mmol/mol	177	1 (0.6)
Fasting insulin >80 pmol/L	175	124 (71)
Serum lipids		
≥1 abnormal lipid	182	79 (43)
Cholesterol >5.2 mmol/L	182	24 (13)
HDL-C <1.0 mmol/L	182	32 (18)
LDL-C >3.4 mmol/L	180	21 (12)
Elevated triglycerides§	182	50 (28)
Liver function tests¶		
≥1 abnormal test	181	85 (47)
AST	149	20 (13)
ALT	181	61 (34)
GGT	181	55 (30)

Abnormal findings based on upper limits of normal. †CRP 1.0–3.0 mg/L moderate risk of future cardiovascular events, >3.0 mg/L <15.0 high risk of future cardiovascular events (using Abbot Architect CRP reagent). ‡Excluding all those with BMI >91st–98th percentile with weight-related co-morbidities (includes *n* = 2 with diabetes). §If aged 0–9 years >1.1 mmol/L, if 10–19 years >1.5.[23] ¶Abnormal if AST and/or ALT and/or GGT > upper limits normal: AST 1 to <7 years, >44 U/L, 7 to <12 years >36 U/L, 12 to <19 years >26 U/L (*n* = 149), ALT 1 to <13 years >25 U/L, 13 to <19 years >22U/L (*n* = 181), GGT 1 to <11 years >16 U/L, 11 to <19 years >21 U/L (*n* = 181).[24] ALT, alanine transaminase; AST, aspartate aminotransferase; ls-CRP, low-sensitivity C-reactive protein; GGT, gamma-glutamyl transferase; HDL-C, high-density lipoprotein cholesterol; HbA1c, glycated haemoglobin; LDL-C, low-density lipoprotein cholesterol.

a device in their bedroom were reported to have difficulty getting to sleep (39% vs. 26%; $P = 0.03$).

Physical examination

The results of physical examinations are summarised in Table 3. Systolic pre-hypertension was present in 4% ($n = 10$), and diastolic pre-hypertension in 5% ($n = 12$), with systolic and diastolic hypertension present in 1% ($n = 2$) and 3% ($n = 8$), respectively.

Investigations

Fasting blood samples were obtained from 183/239 (77%) of participants (Table 4). Average HbA1c was within the normal range (33.8 (SD 3.5) mmol/mol), but there was a high prevalence (71%, $n = 124$) of elevated fasting insulin levels. Low-sensitivity CRP >1 mg/L was found in 137 (75%) of participants, excluding the six with highly elevated low-sensitivity CRP (CRP > 15 mg/L) because of probable acute infection. After adjustment for confounders, higher BMI SDS ($\beta = 0.666$; $P < 0.0001$), WHtR ($\beta = 7.379$; $P < 0.0001$), and WC ($\beta = 0.046$; $P < 0.0001$) were associated with increased low-sensitivity CRP levels, which were in turn associated with higher systolic BP ($\beta = 0.133$; $P = 0.04$).

Ethnic comparisons

Table 5 outlines key parameters in Maori and NZE, excluding those who entered with weight-related comorbidities and diagnoses of type 1 diabetes or type 2 diabetes ($n = 6$). There was a greater prevalence of mothers who smoked during pregnancy ($P = 0.001$) and a family history of type 2 diabetes ($P = 0.02$) among Maori compared with NZE (Table 5). Regarding medical history, Maori had more witnessed breathing pauses than NZE ($P = 0.04$, Table 5). Examination results showed that Maori had a greater average BMI SDS at entry compared with NZE ($P = 0.004$). Acanthosis nigricans was more prevalent in Maori (OR 7.85 (3.89–15.86); $P < 0.0001$), but rates of systolic and/or diastolic pre-hypertension or hypertension were similar in both groups (Table 5).

Amongst laboratory markers, the prevalence of participants with any abnormal serum lipid measure did not differ between Maori and NZE (Table 5). However, Maori were more likely to have low serum high-density lipoprotein cholesterol (27% vs. 14%; OR 2.55(1.10–5.90); $P = 0.03$) and high serum triglyceride (38% vs. 24%; OR 2.18 (1.07–4.43); $P = 0.03$) concentrations than NZE. Maori also had higher fasting insulin concentrations ($P = 0.02$), and were more likely to have a fasting insulin above the upper limit of normal ($P = 0.05$) (Table 5). For liver function tests, the prevalence of any abnormal result was not different in the two ethnic groups (Table 5), but Maori tended to have greater odds of having abnormal gamma-glutamyl transferase results (37% vs. 24%; OR 1.99 (0.98–4.01); $P = 0.056$). Note that when models were adjusted for BMI SDS, ethnic differences in laboratory markers were attenuated and no longer statistically significant.

Table 5 Key parameters among participants of Maori and New Zealand European (NZE) ethnicity at recruitment

	Maori	NZE	*P*-value	*P*-value adjusted for BMI SDS
Family history				
Mother smoking during pregnancy (%)	51 (52)	28 (28)	0.001	—
Type 2 diabetes (%)	70 (71)	56 (55)	0.044	—
Medical history				
Witnessed breathing pauses (%)	27 (25)	15 (14)	0.038	0.17
Examination				
Average BMI SDS, mean (SD)	3.25 (0.62)	3.01 (0.49)	0.004	—
Acanthosis nigricans (%)	62 (58)	21 (20)	<0.0001	<0.0001
Abnormal blood pressure (%)†	13 (12)	10 (9)	0.44	0.93
Laboratory markers‡				
Abnormal fasting lipids (%)§	37 (50)	38 (45)	0.43	0.60
Fasting insulin (pmol/L)	130 (113, 150)	103 (90, 117)	0.015	0.75
Elevated fasting insulin (%)¶	54 (75)	55 (65)	0.051	0.12
Abnormal liver function tests (%)††	38 (52)	37 (44)	0.22	0.58

Fasting insulin data are geometric means and 95% confidence intervals. *P*-values for family history are from χ^2 tests. Other *P*-values are from general linear or logistic regression models, adjusting for age, sex and pubertal status as well as BMI SDS where indicated. †Systolic and/or diastolic pre-hypertension or hypertension. ‡Available for a reduced number of participants (n = 157). §Having any of these parameters: cholesterol >5.2 mmol/L; HDL-C <1.0 mmol/L; LDL-C >3.4 mmol/L; triglycerides: >1.1 mmol/L if aged 0–9 years or >1.5 if 10–19 years.[23] ¶Fasting insulin > 80 pmol/L. ††Abnormal if AST, ALT and/or GGT above the upper limits normal: AST 1 to <7 years, >44 U/L, 7 to <12 years >36 U/L, 12 to <19 years >26 U/L (n = 149), ALT 1 to <13 years >25 U/L, 13 to <19 years >22 U/L (n = 181), GGT 1 to <11 years >16 U/L, 11 to <19 years >21 U/L (n = 181).[24] ALT, alanine transaminase; AST, aspartate aminotransferase; BMI, body mass index; GGT, gamma-glutamyl transferase; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SDS, standard deviation score.

Discussion

In this sample of 239 overweight and obese children presenting to a community-based intervention programme, substantial and generally unrecognised comorbidities were common. The unique aspect of this study was our ability to achieve high representation of Maori and those from most deprived areas in a semi-rural group of children and adolescents, indicating a high level of acceptability for these target groups. Engagement is vital in order to address and manage associated weight-related comorbidities.

While there were some differences between Maori and NZE in the frequency of some comorbidities, the overall clinical picture of obese children and adolescents in our cohort, irrespective of ethnicity, was of concern. Of note, when models accounted for BMI SDS, ethnic differences in laboratory markers were no longer significant.

Comorbidities likely to impact on cardiovascular health were identified frequently in our cohort; especially abnormal lipids and subclinical inflammation. The most common serum lipid pattern in obesity is mild elevation of total cholesterol and LDL-C, low-density lipoprotein cholesterol (LDL-C), with moderate to severe elevation in triglyceride and decrease in high-density lipoprotein cholesterol (HDL-C), a pattern we observed in the participants.[23] Dyslipidaemia, high BP, and obesity are all correlated with an increased risk of atherosclerotic lesions; the extent of these lesions is determined by the number of risk factors present for cardiovascular disease.[25]

Subclinical inflammation has been postulated to play a role in cardiovascular morbidity, as a linking mechanism of obesity with components of the metabolic syndrome and sleep disordered breathing.[26,27] Serum CRP concentration has been shown in children to be correlated with serum fibrinogen and HDL-C concentrations; cardiovascular risk factors implicated in the development of atheroma.[28] The National Health and Nutrition and Examination Survey showed that, in comparison with children with a BMI between the 15th to <85th percentile, an elevated CRP was almost five times as likely to be present in those children with a BMI of ≥95th percentile.[29] Evolution towards the use of high-sensitivity CRP in adults to predict cardiovascular risk has led to high-sensitivity CRP concentrations of <1, 1–3 and >3 mg/L being used as groupings for low-, moderate- and high-risk of future cardiovascular events.[30] Whilst low-sensitivity CRP is not as accurate as high-sensitivity CRP, and the categories are not designed to be used for low-sensitivity CRP, the reality is that high-sensitivity CRP remains unavailable to many in routine paediatric clinical practice. Given the strong associations found between elevated low-sensitivity CRP and multiple cardiovascular risk factors in this cohort, the categories developed for high-sensitivity CRP were adopted here.

Consistent with previous literature,[6,23,31] we showed increased metabolic morbidity including elevated fasting insulin levels, abnormal lipids and type 2 diabetes. Other biochemical features of the metabolic syndrome were also seen with a high frequency of abnormal lipid profiles, liver function tests and elevation in CRP.

Obstructive sleep apnoea is of particular relevance within obese cohorts because of the known associations between obstructive sleep apnoea and cardiovascular morbidity, attention

deficit hyperactivity disorder, learning difficulties and metabolic morbidity.[32] However, the lack of polysomnography in our study meant that we were unable to identify the true prevalence of obstructive sleep apnoea within our cohort. Depending on criteria, approximately 30% of obese children have sleep-related disordered breathing.[33] Whilst 20% of our cohort had witnessed breathing pauses, half of the children and adolescents were found to snore in their sleep for more than half the time, indicating a higher prevalence of symptomatic obstruction.

Whanau Pakari achieved a high level of Maori participation (over 1.5 times the background population rate for Taranaki). Whilst there were subtle ethnic differences noted, Maori had similar obesity-related comorbidities compared with NZE in terms of clinical significance. However, we found a high rate of maternal smoking during pregnancy, and Maori mothers were almost twice (52%) as likely to smoke during pregnancy. National figures in 1990–1991 showed that Maori mothers were three times more likely to smoke than NZE during pregnancy based upon post-natal self-report (68% vs. 23%, respectively).[34] The rate of maternal smoking at birth among mothers in Taranaki was reported to be 20% in 2013 (maternal post-natal self-report).[35] The available NZ data, based on a cohort that was almost all of NZE ethnicity, has shown that in comparison with children whose mothers did not smoke during pregnancy, children of women who did smoke had an increased BMI at age 3 years.[36]

Previous research has highlighted that there is an increased prevalence of early risk factors for obesity in Maori and Pacific infants in NZ.[37] The Early Life Factors study showed that Maori and Pacific infants were twice as likely to have a mother who is obese, and a mother who consumed higher scores on a 'snacks' dietary pattern such as chocolate, sweets, fizzy drinks and crisps. This highlights the intergenerational challenges obesity poses, as well as the importance of intervening at early stages in the life course.[2]

Almost one third of participating children's care givers reported that their child/teen had difficulty getting to sleep, with almost half having some electronic device such as a television, computer or smartphone in their bedroom. The association we observed between a device in a bedroom and reported difficulty getting to sleep was consistent with previous research findings, where the presence of televisions in children's bedrooms is associated with shorter sleep duration and later bedtimes.[38] A recent study has shown that for fourth to seventh graders in the United States, sleeping near a small screen and/or with a television in the bedroom, resulted in increased screen time and was associated with shorter sleep duration.[39] These are important findings, as there is an increased risk of obesity with shorter sleep duration in childhood and adulthood.[40] Whether parents are using devices in bedrooms to manage sleep difficulties, or whether the devices themselves are leading to sleep difficulties remains unclear. Previous NZ data have shown that young children aged 3–7 years who do not get enough sleep are at increased risk of becoming overweight.[41] Whether these associations are indicative of any causative link warrants further research, with more accurate measures of sleep than proxy report, such as those provided by polysomnography.

Our study provides the most comprehensive overview of obese children and adolescent's medical health in NZ to date. The high participation rate by Maori and those most deprived, two of the groups most affected by obesity in NZ,[5] has allowed for a robust comparison between obese Maori and their NZE counterparts. Accurate knowledge of the frequency of the health-related morbidities of obesity in those population groups for whom obesity is more prevalent is essential if obesity services in the future are to achieve appropriateness and acceptability for their target audiences.[11]

A potential weakness of this study was that this was a referred population, and therefore not one that can be generalised to all children in the study region or nationally. Our BP recordings were also likely to be less reliable than ambulatory BP monitoring, which was not feasible in this programme. Another weakness was the inability to draw firm conclusions from our fasting insulin data, given the wide age range of the cohort, and the lack of Tanner pubertal staging by a clinician. Tanner self-report was deemed most appropriate in the home setting, but this has limited our ability to accurately define the prevalence of hyperinsulinaemia in this cohort.

This study has some clear implications for public health policy. In order to address the obesity epidemic, especially in at-risk groups, ideally 'mainstream' services that are appropriate and acceptable for Indigenous people are required, and this can be achieved. It is imperative these services address weight-related comorbidities as well as obesity itself, in order to improve these children's health outcomes. The burden of disease we found in this cohort, if left unaddressed, will be likely to have a considerable impact on these children's health and wellbeing, adding unsustainable pressure on NZ's health system. Along with serious comorbidities, childhood and adolescent obesity is set to contribute to an increased risk of non-communicable diseases, currently the leading cause of death worldwide.[2]

Conclusion

There is an urgent need to address childhood obesity given its global scale. High-risk groups can be reached with appropriately designed services. This semi-rural cohort of children and adolescents demonstrated concerning levels of morbidity, irrespective of ethnicity.

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